Central venous oxygen saturation monitoring in the critically ill patient

Abstract

In the initial treatment of a critically ill patient, blood pressure, heart rate, urine output, and central venous pressure guide resuscitative efforts. Despite normalization of these variables, global tissue hypoxia may still persist and has been implicated in the development of multigorgan failure and increased mortality. Definitive management includes intensive care unit admission, pulmonary artery catheterization using mixed venous oxygen saturation (SvO2), and hemodynamic optimization. In the absence of or before definitive management, hemodynamic optimization can be performed using central venous oxygen saturation (ScvO2) as a surrogate. The physiology, technology, clinical uses, and rationale for ScvO2 monitoring are reviewed, including issues regarding physiologic equivalence to SvO2. The clinical use of ScvO2 monitoring, evidence-based outcome implications, and limitations of ScvO2 monitoring will also be examined.

Abbreviations: DO2 oxygen delivery, EGDT early goal-directed therapy, ScvO2 central venous oxygen saturation, SvO2 mixed venous oxygen saturation

Shock is defined as the presence of global tissue hypoxia secondary to an imbalance between systemic oxygen delivery (DO2) and demands. During initial patient management, physiologic variables such as blood pressure, heart rate, and urine output guide resuscitative efforts. Despite normalization of these variables, significant imbalances between DO2 and demand result in global tissue hypoxia [1,2]. This global tissue hypoxia, if left untreated, leads to anaerobic metabolism, lactate production, and oxygen debt. The magnitude and duration of oxygen debt have been implicated in the development of multisystem organ failure and increased mortality [4-6]. The current review examines the clinical utility of central venous oxygen saturation (ScvO2) as a surrogate for mixed venous oxygen saturation (SvO2) in the care of critically ill patients.

Physiology of mixed venous oxygen saturation

The normal range for SvO2, which reflects the balance between DO2 and demands, is 65 to 75% [7,8]. SvO2 decreases when DO2 has been compromised or systemic oxygen demands have exceeded supply. When this compensatory mechanism is overwhelmed and SvO2 remains low, global tissue hypoxia and lactic acidosis ensues. SvO2 has been used for its prognostic significance as a predictor of hyperlactemia and death (compared with arterial oxygen saturation and cardiac output In patients with severe cardiac and pulmonary disease) [7,9]. The objective of the clinician is to determine which of these variables are deranged and to institute appropriate therapy. Figure 1 illustrates the variables that affect SvO2.
Figure 1. Variables that affect mixed venous oxygen saturation

Central or mixed venous oxygen saturation can be decreased as a result of variables that increase systemic oxygen consumption or a decrease in systemic oxygen delivery. DO₂, systemic oxygen delivery; Hg, mercury; PaO₂, arterial oxygen pressure; VO₂, systemic oxygen consumption.

Technology of venous oximetry

By using infrared oximetry, which is based on reflection spectrophotometry, Svo₂ can be monitored continuously. Light is transmitted into the blood, reflected off red blood cells, and read by a photo detector. The amount of light reflected at different wavelengths varies depending on the concentration of oxyhemoglobin and hemoglobin (Fig. 2). The catheter used to measure venous oxygen saturation can be a pulmonary artery catheter or a 16 to 22 cm central venous catheter capable of fluid or medication administration and pressure measurements.

Figure 2. The technology of spectrophotometry

The technology of spectrophotometry involves sending an infrared signal by fiberoptic transmission through the central venous catheter. A receiving fiber in the catheter detects reflected light off of hemoglobin in red blood cells to provide a continuous read-out of central venous oxygen saturation.

Clinical uses of mixed venous oxygen saturation monitoring

Used extensively in various clinical scenarios, Svo₂ has been shown to be superior to mean arterial pressure and heart rate in...
cardiac surgery patients [10]. Declines in SvO₂ preceded the onset of inadequate myocardial function [11], shock, or the development of arrhythmias, although vital signs were normal [12]. SvO₂ has been shown to have diagnostic, prognostic, and therapeutic use in the treatment of critically ill patients who have acute myocardial infarction [13,14] or general medical intensive care unit (ICU) conditions [15]; have undergone postoperative cardiovascular procedures [16••], vascular surgery [20,21], pediatric surgery [11], or lung transplantation [26]; have experienced trauma [17–19], septic shock [22,23], or cardiogenic shock [24,27]; or are neonates [25]. Although SvO₂ has not been shown to have outcome benefit as a goal-directed hemodynamic end-point in a heterogeneous group of patients after ICU admission [28], it has shown to decrease morbidity and health care-resource consumption in postoperative cardiovascular [16••] and trauma [17] patients. There is evidence that the timing of diagnostic and therapeutic intervention using this technology may be a critical outcome determinant that has not been addressed in prior studies [29••].

Why monitor central venous oxygen saturation?

The time between the onset of critical illness and definitive ICU intervention can be significantly long and have outcome implications [30,31••,32]. Measurement of SvO₂ requires placement of a pulmonary artery catheter, which may not be feasible early in resuscitation or in pediatric patients. However, central venous oxygen saturation can be obtained in both ICU and non-ICU settings, which makes continuous ScvO₂ monitoring a convenient surrogate for SvO₂. Up to 50% of patients resuscitated from shock may have continued global tissue hypoxia (i.e., increased lactate and decreased ScvO₂) even with the normalization of vital signs and central venous pressure [3••]. The ability to detect and resolve occult tissue hypoxia early in the course of patient care may have outcome benefit [16••,29••].

Are mixed central venous oxygen saturation clinically equivalent?

The relation between SvO₂ and ScvO₂ obtained from the superior vena cava and right atria has been examined in animal and human models (Table 1). Superior vena cava ScvO₂ is slightly less than right atria ScvO₂ and more accurately reflects SvO₂ when patients are not in shock [33,34••]. Right atria saturations are not significantly different from SvO₂ whether or not in shock [33]. For patients in shock a consistent reversal of this relation occurs and superior vena cava ScvO₂ is always greater than SvO₂ [33]; the difference can range from 5 to 18% [33,34••]. Redistribution of blood flow away from the splenic, renal, and mesenteric bed toward the cerebral and coronary circulation, including more desaturated blood (<30%) from the coronary sinus, contributes to this observation [33]. Thus, superior vena cava ScvO₂ consistently overestimates the true SvO₂ under shock conditions.
Table 1. Studies comparing mixed venous oxygen saturation and central venous oxygen saturation.

CPR, cardiopulmonary resuscitation; PA, pulmonary artery; ScvO₂, central venous oxygen saturation; SvO₂, mixed venous oxygen saturation.
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<td>Kong et al.</td>
<td>Patients with end-stage renal failure</td>
<td>Cardiac index was lower in patients 2.45 ± 0.42 L/min/m² compared with the control subjects 3.74 ± 0.17 L/min/m². SvO₂ was 53 ± 8% and ScvO₂ (superior vena cava) was 57 ± 6%.</td>
<td>Venous oxygen saturations are similar in end-stage renal failure.</td>
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<td>Wendt et al.</td>
<td>Patients in the ICU.</td>
<td>The correlation of oxygen partial pressures was 0.687 and the correlation of the saturation reached 0.779. The calculation of venous admixture showed a correlation of 0.901.</td>
<td>ScvO₂ adequately reflects ScvO₂.</td>
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<td>Emeran et al.</td>
<td>Experimental cardiac arrest dog model.</td>
<td>PO₂, PCO₂, and pH from the pulmonary artery samples were strongly correlated with those from the central venous (r = 0.93, 0.93, and 0.93, respectively) and from the femoral venous samples (r = 0.73, 0.93, and 0.97, respectively). There were no significant differences in the pulmonary artery, central, or femoral venous gases.</td>
<td>Femoral and central venous samples mirror true mixed venous blood gases from the PA and could be used in their place.</td>
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<td>Martin et al.</td>
<td>Continuous monitoring in critically ill patients with and without therapeutic interventions</td>
<td>Systematic error was 0.6% and 3% and variability was 10%. Differences were greater than or equal to 5% in 49% of values during periods of stability and in 50% of values during periods with therapeutic interventions. Correlation was r = 0.48 without and r = 0.62 with therapeutic interventions. Changes in ScvO₂ and SvO₂ during periods without and with therapeutic interventions were r = 0.70 and r = 0.77, respectively.</td>
<td>The present study indicates that ScvO₂ monitoring was not reliable in the study patients.</td>
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<td>Martin et al.</td>
<td>Open-chest CPR animal model.</td>
<td>The correlation between ScvO₂ and SvO₂ during CPR with values ranging from 0.1569 (P = 0.542) to 0.5781 (P = 0.024). Although statistically significant at times, the correlation between ScvO₂ and SvO₂ during CPR is not consistently high enough to enable the routine substitution in this model.</td>
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CPR, cardiopulmonary resuscitation; ICU, intensive care unit; PA, pulmonary artery; ScvO₂, central venous oxygen saturation; SvO₂, mixed venous oxygen saturation.

Table 1. Studies comparing mixed venous oxygen saturation and central venous oxygen saturation (continued)

There has been considerable debate regarding whether ScvO₂ is a satisfactory substitute for SvO₂, particularly in ranges above 65%[35••,36••-39••]. Although the absolute values of ScvO₂ and SvO₂ differ, studies have shown close tracking of the two sites across a wide range of hemodynamic conditions [40••]. Furthermore, the presence of a pathologically low ScvO₂ value (implying an even lower SvO₂) is more clinically important than whether the values are equal. Goldman et al. [13] found that ScvO₂ less than 60% showed evidence of heart failure, shock, or a combination of the two. Hyperdynamic septic shock ICU patients seldom exhibit SvO₂ levels less than 60 to 65%, and sustained levels are associated with increased mortality [22,23]. Studies examining the clinical utility of ScvO₂ early in the course of disease presentation routinely encounter values less than 50%, which are considered critical [3,29•••,41••]. At these values, venous saturations are actually 5 to 18% lower in the pulmonary artery [33,42] and 15% lower in the splanchic bed [43]. Thus, although not numerically equivalent, these range of values are pathologically equivalent [13] and associated with high mortality [9].

Clinical uses of central venous oxygen saturation monitoring

Rady et al. [3••] found that 50% of critically ill patients presenting in shock who were resuscitated to normal vital signs continued to have increased lactate and abnormally low ScvO₂, indicating anaerobic metabolism and oxygen debt. These patients required further interventions. Rady et al. ’s study gave rise to the clinical use of ScvO₂ in early management of cardiac arrest, the postresuscitation period, trauma and hemorrhage, severe heart failure, severe sepsis, and septic shock (Table 2).
Cardiac arrest

Management of the cardiac-arrest patient by advanced cardiac life support guidelines includes physical examination (fe, palpation of a pulse) and electrocardiographic monitoring. ScvO₂ monitoring during cardiac arrest has been shown to be a diagnostic and therapeutic adjunct [44-46]. Cardiac-arrest patients routinely have ScvO₂ values of 5 to 20% during cardiopulmonary resuscitation. Failure to reach ScvO₂ of at least 40% during the management of cardiac arrest is associated with a 100% mortality rate even when there is intermittent measurable blood pressure. ScvO₂ has been used to confirm the presence or absence of sustainable cardiac activity during electromechanical dissociation (EMD) or a pulseless idioventricular rhythm (where over 35% of patients have been shown to have spontaneous cardiac activity [pseudo-EMD]) [47]. If ScvO₂ is greater than 60%, return of spontaneous circulation (ROSC) is likely and the pulse should be frequently rechecked if EMD is present. Between ScvO₂ values of 40% and 72%, there is a progressive increase in the rate of ROSC. When ScvO₂ greater than 72% is obtained, ROSC has likely occurred. Continuous ScvO₂ monitoring provides an objective measure to confirm the adequacy or inadequacy of cardiopulmonary resuscitation in providing DO₂.
Postresuscitation after cardiac arrest
In the immediate postresuscitation period, patients are frequently hemodynamically unstable and have a high frequency of re-arrest. Cuff and intraarterial pressures may be rendered insensitive in the measurement of cardiac output or DO₂ secondary to the high systemic vascular resistance of catecholamine therapy [1,45,48]. An abrupt or gradual decrease in ScvO₂ (< 40-50%) indicates likelihood for re-arrest, whereas ScvO₂ greater than 60 to 70% indicates hemodynamic stability. A sustained extreme elevation of ScvO₂ (> 80%) in the presence of a low DO₂ carries a poor prognosis because it indicates an impairment of systemic oxygen consumption (ie, the inability of the tissues to use oxygen). This has been attributed to long periods of arrest and the use of large doses of vasopressors [49]. If this derangement is not corrected within the early postresuscitation period, the outcome is uniformly fatal [45].

Traumatic and hemorrhagic shock
The standards of Advanced Trauma Life Support focus on normalization of vital signs [50]. Studies have shown that vital signs are insensitive end-points of resuscitation and outcome predictors in hemorrhagic and trauma resuscitation [1,51]. Kowalenko et al. [52] and Scalea et al. [51] have shown that patients presenting with trauma and hemorrhage required additional resuscitation or surgical procedures when ScvO₂ remained less than 65%.

Occult cardiogenic shock in severe heart failure
Cardiogenic shock is characterized by decreased DO₂ and evidence of tissue hypoxia (eg, lactic acidosis, end-organ dysfunction) secondary to myocardial dysfunction [24]. This definition is most commonly seen in acute pump dysfunction (eg, acute myocardial infarction). In chronic severe heart failure, this presentation may be insidious. Ander et al. [41] examined patients who presented with decompensated chronic severe heart failure (ejection fraction < 30%) who were stratified into normal and elevated lactate (> 2 mm/L) groups. There was a significant prevalence of “occult cardiogenic shock” with ScvO₂ ranging from 26.4 to 36.8% in the presence of normal vital signs. Using a goal-oriented approach of preload, afterload, contractility, coronary perfusion, and heart-rate optimization, these patients required additional therapy whereas their counterparts with normal lactate levels did not [41].

Severe sepsis and septic shock: an outcome evaluation of early intervention
Previous studies have examined Svo₂-guided goal-directed therapy for severe sepsis and septic shock after ICU admission. In a study evaluating early goal-directed therapy (EGDT) using ScvO₂ before ICU admission, patients presenting with severe sepsis and septic shock were randomized to 6 hours of EGDT or standard therapy before ICU admission. Patients in both groups were resuscitated to a central venous pressure greater than 8 mm Hg and mean arterial pressure greater than 65 mm Hg, but those in the treatment group were resuscitated to an ScvO₂ greater than 70% using continuous ScvO₂ monitoring. Serial end-points of resuscitation, physiologic and organ dysfunction scores, mortality rate, and health care-resource consumption were compared at 0, 6, 12, 24, 36, 48, 60, and 72 hours.

There were no significant differences in 0-hour variables between the EGDT and control group patients. Over the initial 72 hours, there was a higher central venous oxygen saturation (65.4 ± 8.9% vs 57.1 ± 13%, P < 0.001), lower lactate (3.1 ± 3.0 mmol/L vs 4.0 ± 3.8 mmol/L, P = 0.025), lower base deficit (2.4 ± 4.6 mEq/L vs 5.6 ± 6.0 mEq/L, P < 0.001), and higher pH (7.39 ± 0.20 vs 7.35 ± 0.14, P = 0.01) for those in the EGDT group than for those in the control group. Values for Acute Physiology and Chronic Health Evaluation Score-II, Simplified Acute Physiology Score-II, and Multiple Organ Dysfunction Syndrome were lower for patients in the EGDT group at all time points than they were for those in the control group (all P < 0.001). In-hospital mortality was 26.1% versus 42.4% (P = 0.009) and 28-day mortality was 30.5% versus 48.2% (P = 0.008) for those in the EGDT group versus the control group, respectively. In survivors, duration of hospital stay and mechanical ventilation were 3.8 (P = 0.001) and 1.4 days (P = 0.001) less in the EGDT group than in the control group, respectively. Using ScvO₂ as a resuscitation end-point in addition to mean arterial pressure and central venous pressure provides significant outcome benefit for patients with severe sepsis and septic shock over standard therapy.

Limitations and future questions
Despite studies questioning the value of Svo₂ in treating ICU patients [45,53-55], considerable evidence suggests that ScvO₂ has a beneficial role in the early management of critically ill patients and even neonates [25,56]. The ability to access this information earlier in the phases of critical illness is now a reality and further studies are now in progress to confirm that early recognition and treatment of deranged ScvO₂ values have significant outcome benefit.

Conclusions
In the resuscitation of critically ill patients, ScvO₂ monitoring has been shown to be a better indicator of tissue oxygenation and derangement of cellular oxygen utilization than vital signs. When applied to the treatment of various shock states, ScvO₂ monitoring impacts therapeutic intervention; there is evidence that this may be of outcome benefit over conventional therapy, which uses vital signs and central venous pressure.
References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

• Of special interest
•• Of outstanding interest


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